comprehensive outpatient care program to minimize the incidence of mortality, morbidity and admittance to hospital. Proper selection and education of patients as well as follow-up analyses will help to avoid financial waste, unrealistic expectations and poorly coordinated treatment regimens.

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Computed Tomography in Lung Disease

COMPUTED TOMOGRAPHIC (CT) scanning in patients with lung disease has proved useful in (1) detection and evaluation of solitary pulmonary nodules, (2) detection of pulmonary metastasis, (3) diagnosis and evaluation of vascular lung lesions, (4) distinguishing between pulmonary parenchymal and pleural processes, (5) the detection and evaluation of diffuse pulmonary disease and (6) carrying out percutaneous needle aspiration biopsy.

In general, CT is more sensitive than are plain radiographs or conventional tomograms in the detection of single or multiple pulmonary nodules. For this reason, CT is largely replacing conventional tomographic studies for the detection of nodular disease. In patients with a solitary nodule visible on plain radiographs, CT can be used to search for other nodules or associated mediastinal adenopathy; CT is also more sensitive in detecting mediastinal lesions. CT densitometry of solitary nodules may be of some value in distinguishing benign and malignant lesions, the presumption being that benign lesions often contain calcium invisible on plain radiographs but detectable by CT. In one study, excellent results were obtained by measuring the CT number of lung nodules detected. However, these results have been difficult to duplicate, and CT nodule densitometry, at this time, must be considered experimental or of limited clinical value.

Because of its enhanced sensitivity and ability to view the mediastinum, CT should replace other radiographic procedures in the detection of pulmonary metastasis in most cases.

Dynamic CT scanning following a bolus injection of contrast material can be used to diagnose and evaluate pulmonary vascular lesions, such as arteriovenous fistula, pulmonary vein varix and sequestration. However, vascular tumors (bronchial adenoma) sometimes may produce false-positive results. In some cases, CT may replace arteriography.

Because of its cross-sectional format, CT is ideally suited to the distinction of pulmonary parenchymal disease and pleural disease when plain chest radiographs are equivocal. However, although CT has been reported helpful in differentiating empyema and lung abscess, this distinction can be difficult.

CT is extremely sensitive in detecting subtle differences in density and can show pulmonary fibrosis in patients with various lung diseases (such as sarcoidosis or asbestosis) when plain chest radiographs show no abnormalities. Also, CT shows areas of decreased density (such as bullae or emphysema) better than other radiographic techniques.

Lastly, in patients with pulmonary lesions requiring percutaneous biopsy, CT can be extremely useful in needle placement and precise localization of the needle tip. By using CT, lesions in the lung that are close to vascular structures can be safely approached.

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Diagnosis and Management of Goodpasture's Syndrome

GOODPASTURE'S SYNDROME involves a triad of glomerulonephritis, pulmonary hemorrhage and antiglomerular basement membrane antibody (anti-GBM) production. The cause of this disorder is unknown, although some patients have a history of recent viral illness or exposure to volatile hydrocarbons. It is an uncommon but serious disorder in which a rapidly progressive, crescentic glomerulonephritis develops, which leads to uremia. Pulmonary hemorrhage in the syndrome may be life-threatening, and evidence of its presence—that is, hemoptysis, infiltrates noted on x-ray films, reduced arterial oxygen tension, hemosiderin-laden macrophages and iron deficiency anemia—is

found in 50 percent to 75 percent of cases. Increased pulmonary carbon monoxide uptake is present in nearly all patients, and is a sensitive test of pulmonary hemorrhage.

Goodpasture's syndrome must be distinguished from other causes of glomerulonephritis and pulmonary hemorrhage such as systemic lupus erythematosus or necrotizing vasculitis where tissue injury is mediated by immune complexes. Also, in patients with established renal failure, pulmonary hemorrhage caused by pneumonia, pulmonary emboli or a bleeding diathesis can develop. Accurate diagnosis depends on detection of circulating anti-GBM antibody, present in more than 90 percent of patients, by indirect immunofluorescence or radioimmunoassay and by kidney or lung biopsy and the demonstration of *linear* IgG deposits along the basement membrane. Absence of such *linear* IgG deposits in a kidney biopsy excludes the diagnosis of Goodpasture's syndrome. However, the reliability of lung biopsy findings has not been established and a negative study may not be definitive. Circulating anti-GBM antibody levels do not correlate with severity of clinical manifestations. If properly done, examination of serum for anti-GBM antibodies rarely gives false-positive results. While *linear* IgG deposits along the GBM in the kidney also occurs in diabetes and occasionally in other conditions, the use of proper immunofluorescence controls can usually aid in distinguishing between these conditions.

High doses (10 to 20 mg per kg of body weight) of methylprednisolone ("pulse therapy") and intensive plasma exchange (plasmapheresis) appear to represent important new treatments for pulmonary hemorrhage in Goodpasture's syndrome and have largely replaced the questionable practice of bilateral nephrectomy for life-threatening disease. Plasmapheresis will quickly reduce the level of circulating anti-GBM antibody. For severe pulmonary hemorrhage unresponsive to "pulse therapy," daily plasma exchanges (3 to 4 liters) may be done using semiautomated, gravity-sedimentation (centrifugation) techniques for separating formed elements from plasma. The removed plasma may be replaced with plasma protein fractions or fresh frozen plasma. Plasma exchange is combined with oral administration of prednisone (1 mg per kg of body weight per day), cyclophosphamide (2 mg per kg of body weight per day) and, in patients younger than 50, azathioprine (1 to 2 mg per kg of body weight per day). Daily or every-other-day exchanges are continued until there is clear evidence of clinical and laboratory improvement, and the drugs are administered for two to six months or longer. In most patients cessation of pulmonary hemorrhage occurs within 24 to 72 hours of initiating plasma exchange treatment. If renal failure is not advanced (serum creatinine <6 mg per dl), renal function often stabilizes or improves. Infection must be scrupulously avoided because it may exacerbate the pulmonary hemorrhage and renal failure.

Surprisingly, anti-GBM antibody levels do not parallel the improvement and should not be used as a sole guide for treatment. Plasmapheresis is not a substitute for hemodialysis and both procedures may be carried out concurrently. Patients with advanced renal failure (serum creatinine levels greater than 10 mg per dl) and severe glomerular and tubulointerstitial lesions may not experience any improvement in renal function and may require maintenance hemodialysis. Renal transplantation is not contraindicated but should be deferred until anti-GBM antibody levels have decreased to low or undetectable levels. Recurrence of disease in the allograft may occur but usually is not severe enough to result in graft failure.

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Increased-Permeability Edema of the Lung

OF THE TWO CHIEF TYPES of acute pulmonary edema—that due to increased pressure and that due to increased alveolar capillary barrier permeability (formerly called noncardiogenic edema)—the latter has been the focus of much clinical and experimental attention for three reasons: its rapid course, its development in otherwise healthy persons and an emerging understanding of its pathophysiology.

The chief clinical characteristics of increased-permeability edema are as follows: a specific inciting event, rapid time course, high edema fluid to plasma protein concentration ratio and pulmonary artery wedge pressure usually within the broad range of normal. Wedge pressure elevation does